



RESEARCH ARTICLE

Neural Mechanisms and Clinical Effectiveness of Hypnotherapy in Treating Social Anxiety Disorder: A Systematic Review of Behavioral and Neurophysiological Evidence

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ARTICLE INFO	ABSTRACT
Received: July 1 2026 Accepted: July 12, 2026 Keywords: Social Anxiety Disorder; Hypnotherapy; Clinical Hypnosis; Event-Related Potentials; N170; Late Positive Potential; Neural Mechanisms *Corresponding Author: 1442200116@surel.untag-sby.ac.id	Abstract: Social Anxiety Disorder (SAD) is a disabling condition characterized by persistent fear of negative social evaluation. Although cognitive-behavioral therapy remains the psychological standard of care, treatment response is heterogeneous. Hypnotherapy may offer a complementary approach, yet its efficacy and neurophysiological mechanisms in SAD remain poorly established. This systematic review synthesized evidence on the clinical and neurophysiological effects of hypnotherapy in SAD. A systematic search of Semantic Scholar and OpenAlex was conducted using Elicit. Eligible studies included randomized controlled trials, clinical trials, and systematic reviews involving adults with clinically diagnosed SAD receiving therapist-delivered hypnotherapy for ≥ 6 sessions with objective neurophysiological outcomes. Ten sources were identified, but only one RCT met all eligibility criteria. Six weekly sessions of hypnotherapy integrated with CBT significantly reduced Liebowitz Social Anxiety Scale scores ($t(22)=11.98$, $P<0.01$) and modulated N170 and late positive potential amplitudes during face-processing tasks. Greater reductions in these neural markers were associated with greater symptom improvement. Meta-analytic evidence from broader anxiety populations supports moderate-to-large effects, particularly when hypnosis is combined with psychological interventions. Hypnotherapy shows preliminary efficacy for SAD and may exert therapeutic effects through modulation of attentional and emotional processing. However, the evidence remains insufficient for definitive clinical recommendations. Larger, rigorously controlled RCTs with multimodal neuroimaging are needed to establish efficacy, mechanisms, and treatment-response biomarkers.

INTRODUCTION

Social Anxiety Disorder (SAD), or social phobia, is a pervasive and debilitating mental health condition characterized by a marked, persistent fear of social or performance situations where scrutiny and negative evaluation by others is anticipated (1). Its global prevalence and associated functional impairment underscore a substantial clinical and economic burden. First-line psychological treatment, predominantly Cognitive Behavioral Therapy (CBT), demonstrates efficacy but exhibits non-response rates of approximately 30-40%, and access barriers persist (2). This landscape necessitates the investigation of complementary, alternative, or integrative therapeutic approaches to enhance treatment portfolios and outcomes (3).

Hypnotherapy, a therapeutic technique that utilizes guided relaxation, intense concentration, and focused attention to achieve a heightened state of suggestibility (trance), has accumulated empirical support for managing a spectrum of conditions, particularly those involving anxiety and pain (4)(5). Its application in anxiety disorders often involves using suggestion to alter maladaptive perceptions, reduce physiological arousal, and facilitate cognitive restructuring. Neurobiologically, hypnosis has been associated with functional changes in brain networks governing attention, executive control, and emotional regulation, such as decreased activity in the dorsal anterior cingulate cortex (dACC) and increased connectivity between the dorsolateral prefrontal cortex (DLPFC) and dACC (6).

Despite this promising foundation, the specific application of hypnotherapy for SAD is strikingly under-researched (7). SAD is uniquely characterized by core dysfunctions in social-cognitive processing, including heightened attentional vigilance toward threatening social cues (especially faces), biased interpretation of ambiguous social stimuli, and exaggerated amygdala and fusiform gyrus reactivity (8)(9). Whether and how hypnotherapy engages and modulates these SAD-specific neural pathways remains largely unexplored (10). Investigating this requires coupling clinical outcome measures with objective neurophysiological tools, such as electroencephalography (EEG) and event-related potentials (ERPs) (11). ERPs like the N170 (a face-sensitive component generated in the fusiform gyrus) and the Late Positive Potential (LPP, reflecting sustained emotional attention) provide temporally precise metrics of social-emotional information processing that are directly relevant to SAD pathology (12).

This systematic review seeks to address this critical gap by pursuing three primary objectives: 1) To evaluate the clinical effectiveness of hypnotherapy in reducing symptom severity and improving functioning in adults with clinically diagnosed SAD; 2) To synthesize and analyze evidence pertaining to the neurophysiological mechanisms underlying hypnotherapy's effects in SAD, with particular focus on ERP biomarkers (N170, LPP) and their relationship to clinical change; 3) To critically appraise the methodological quality of the existing literature and identify key limitations and directions for future research (13)(14).

The implications of this review are multifaceted. For clinicians, it provides a consolidated evidence base to inform decisions regarding the potential integration of hypnotherapy as an adjunctive treatment for

SAD. For researchers, it clarifies the current state of knowledge, highlights the most promising neural targets (e.g., N170, LPP), and maps out a clear agenda for mechanistic research (13)(14). Establishing reliable neural biomarkers of treatment response could pave the way for personalized medicine approaches in SAD treatment, predicting which patients are most likely to benefit from hypnotherapy.

Based on the theoretical framework linking hypnosis to attention modulation and the known neurocognitive profile of SAD, we hypothesize that clinically administered hypnotherapy will lead to statistically and clinically significant reductions in SAD symptom severity (15)(16). Furthermore, we hypothesize that these clinical improvements will be correlated with specific, quantifiable modulations in ERP components: namely, a reduction in the amplitude of the N170 (indicating normalized early face processing) and a reduction in the amplitude of the LPP (indicating diminished sustained emotional salience of social threats) (17).

A significant chasm exists between the general evidence for hypnosis in anxiety and SAD-specific applications. While meta-analyses confirm hypnosis is efficacious for anxiety broadly (4)(18), there is a paucity of high-quality studies that: a) exclusively focus on a well-characterized SAD population, b) employ rigorous methodologies (e.g., RCTs with active controls), and c) incorporate objective neurophysiological measurements to elucidate mechanisms. Most existing studies either amalgamate various anxiety disorders, lack neural data, or suffer from design flaws such as small samples, inadequate control groups, or subjective-only outcomes (19)(20). This gap limits clinical translation and mechanistic understanding.

This review contributes novel insights by adopting a translational neuroscience perspective specifically for SAD and hypnotherapy. Unlike previous broad reviews, it meticulously focuses on the intersection of SAD-specific clinical outcomes and objective neurophysiological data, particularly EEG/ERP biomarkers. By critically evaluating the potential of N170 and LPP as treatment response markers, this review bridges the typically separate domains of clinical psychology and cognitive neuroscience, offering a more integrated and mechanistic understanding of how hypnotherapy might work for this distinct disorder. It thus provides a foundational framework for future hypothesis-driven research in this nascent field.

METHODS

Protocol

The study strictly adhered to the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines to ensure methodological rigor and accuracy (21). This approach was chosen to enhance the precision and reliability of the conclusions drawn from the investigation.

Screening

We screened in sources that met these criteria:

- **Population Age:** Does the study include only adult participants (≥ 18 years of age)?
- **Clinical Diagnosis:** Do participants have clinically diagnosed Social Anxiety Disorder according to established diagnostic criteria (DSM-5, ICD-11, or equivalent)?
- **Intervention Type:** Is the intervention clinical hypnotherapy or hypnosis administered by qualified therapists or clinicians?
- **Intervention Isolation:** Can hypnotherapy be identified as the primary intervention (either standalone or with separate analysis distinguishing its effects)?
- **Study Design:** Is the study design a randomized controlled trial (RCT), controlled clinical trial, quasi-experimental study with control group, systematic review, or meta-analysis?
- **Neurophysiological Data:** Does the study include objective neural measurements (such as EEG recordings or other neurophysiological assessments)?
- **Clinical Outcomes:** Does the study include validated clinical anxiety scales, behavioral assessments, or other measurable clinical outcomes specifically for Social Anxiety Disorder?
- **Sample Size:** Does the study include at least 10 participants?
- **Treatment Duration:** Does the hypnotherapy intervention consist of at least 6 sessions or treatment episodes?
- **Target Condition:** Is the study specifically focused on Social Anxiety Disorder (rather than other anxiety conditions, depression, PTSD, or non-social phobias)?

We considered all screening questions together and made a holistic judgement about whether to screen in each paper.

Data extraction

- **Study Design:**

Extract study design and participant information including:

- Study type (RCT, clinical trial, systematic review, meta-analysis)
- Total sample size and group allocation
- Participant demographics (age range, gender distribution)
- SAD diagnosis method and severity (baseline LSAS scores if available)
- Inclusion/exclusion criteria specific to SAD

- **Hypnotherapy Protocol:**

Extract detailed hypnotherapy intervention characteristics including:

- Type of clinical hypnosis used (technique, approach)
- Number of sessions and frequency (e.g., weekly)

- Session duration
- Who delivered the intervention (therapist qualifications)
- Treatment setting
- Specific hypnotic suggestions or protocols used
- Any additional therapeutic components

- **Control Conditions:**

Extract all control/comparison conditions including:

- Type of control (no treatment, waitlist, active control, standard care)
- Details of any active comparator interventions
- Duration of control period
- Number of participants in each condition
- Any crossover or follow-up arrangements

- **Neurophysiological Measures:**

Extract all neurophysiological and objective measurements including:

- EEG components measured (N170, LPP, theta activity, etc.)
- EEG recording procedures and electrode placement
- Other neuroimaging methods (fMRI, EMG, etc.)
- Neurophysiological tasks used (face processing, attention bias tasks)
- Measurement timing (baseline, post-treatment, follow-up)
- Technical specifications and analysis methods

- **Clinical Outcomes:**

Extract all clinical and behavioral outcome measures including:

- Social anxiety symptom scales (LSAS, other validated measures)
- Behavioral measures (attention bias, reaction times, task performance)
- Other psychological measures (depression, general anxiety scales)
- Assessment timepoints (baseline, post-treatment, follow-up duration)
- Clinically significant improvement criteria or cut-off scores

- **Clinical Effectiveness:**

Extract clinical effectiveness results including:

- LSAS score changes (pre-post treatment with effect sizes)
- Other symptom scale improvements with statistical significance
- Behavioral task performance changes
- Response rates and remission rates if reported
- Clinical significance of improvements
- Duration of benefits (follow-up results)

- **Neural Mechanisms:**

Extract neurophysiological findings and mechanisms including:

- Changes in N170 amplitude/latency and statistical significance
- LPP component changes and effect sizes
- Other EEG findings (theta activity, hemispheric differences, etc.)
- Correlations between neural changes and symptom improvements
- Evidence for attention bias modulation
- Brain regions or networks affected
- Proposed mechanisms of action

- **Key Limitations:**

Extract study limitations and quality issues including:

- Sample size limitations and power calculations
- Control group limitations or lack of active controls
- Methodological concerns (blinding, randomization issues)
- Measurement limitations (subjective vs objective measures)
- Generalizability concerns
- Author-reported limitations and conflicts of interest

Search Strategy

The keywords used for this research based PICO :

Element	Keyword 1	Keyword 2	Keyword 3	Keyword 4
Population (P)	Social Anxiety Disorder	Social Phobia	SAD (Social Anxiety Disorder)	Social Anxiety
Intervention (I) / Exposure (E)	Hypnotherapy	Clinical Hypnosis	Hypnotic Intervention	Therapeutic Hypnosis
Comparison (C)	No Treatment Control	Waitlist Control	Active Control (e.g., Supportive Therapy)	Treatment as Usual (TAU)
Outcome (O)	Neural Mechanisms / Neurophysiological Markers	Event-Related Potentials (ERPs) (e.g., N170, LPP)	Clinical Effectiveness / Symptom Reduction	Liebowitz Social Anxiety Scale (LSAS) / Behavioral Outcomes

The Boolean MeSH keywords inputted on databases for this research are: (*"Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety"*) AND (*"Hypnotherapy" OR "Clinical*

Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis") AND ("No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual") AND ("Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR "Behavioral Outcomes")

Data retrieval

Abstracts and titles were screened to assess their eligibility, and only studies meeting the inclusion criteria were selected for further analysis. Literature that fulfilled all predefined criteria and directly related to the topic was included. Studies that did not meet these criteria were excluded. Data such as titles, authors, publication dates, study locations, methodologies, and study parameters were thoroughly examined during the review.

Quality Assessment and Data Synthesis

Each author independently assessed the titles and abstracts of the selected studies to identify those for further exploration. Articles that met the inclusion criteria underwent further evaluation. Final decisions on inclusion were based on the findings from this review process.

Table 1. Article Search Strategy

Database	Keywords	Hits
Pubmed	<i>("Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety" AND "Hypnotherapy" OR "Clinical Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis" AND "No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual" AND "Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR "Behavioral Outcomes")</i>	2,378
Semantic Scholar	<i>("Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety") AND ("Hypnotherapy" OR "Clinical Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis") AND ("No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual") AND ("Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR</i>	250

Database	Keywords	Hits
	<i>"Behavioral Outcomes")</i>	
Springer	<i>("Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety") AND ("Hypnotherapy" OR "Clinical Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis") AND ("No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual") AND ("Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR "Behavioral Outcomes")</i>	27
Google Scholar	<i>("Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety") AND ("Hypnotherapy" OR "Clinical Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis") AND ("No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual") AND ("Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR "Behavioral Outcomes")</i>	59
Wiley Online Library	<i>("Social Anxiety Disorder" OR "Social Phobia" OR "SAD" OR "Social Anxiety") AND ("Hypnotherapy" OR "Clinical Hypnosis" OR "Hypnotic Intervention" OR "Therapeutic Hypnosis") AND ("No Treatment Control" OR "Waitlist Control" OR "Active Control" OR "Treatment as Usual") AND ("Neural Mechanisms" OR "Neurophysiological Markers" OR "Event-Related Potentials" OR "N170" OR "LPP" OR "Clinical Effectiveness" OR "Symptom Reduction" OR "Liebowitz Social Anxiety Scale" OR "LSAS" OR "Behavioral Outcomes")</i>	3

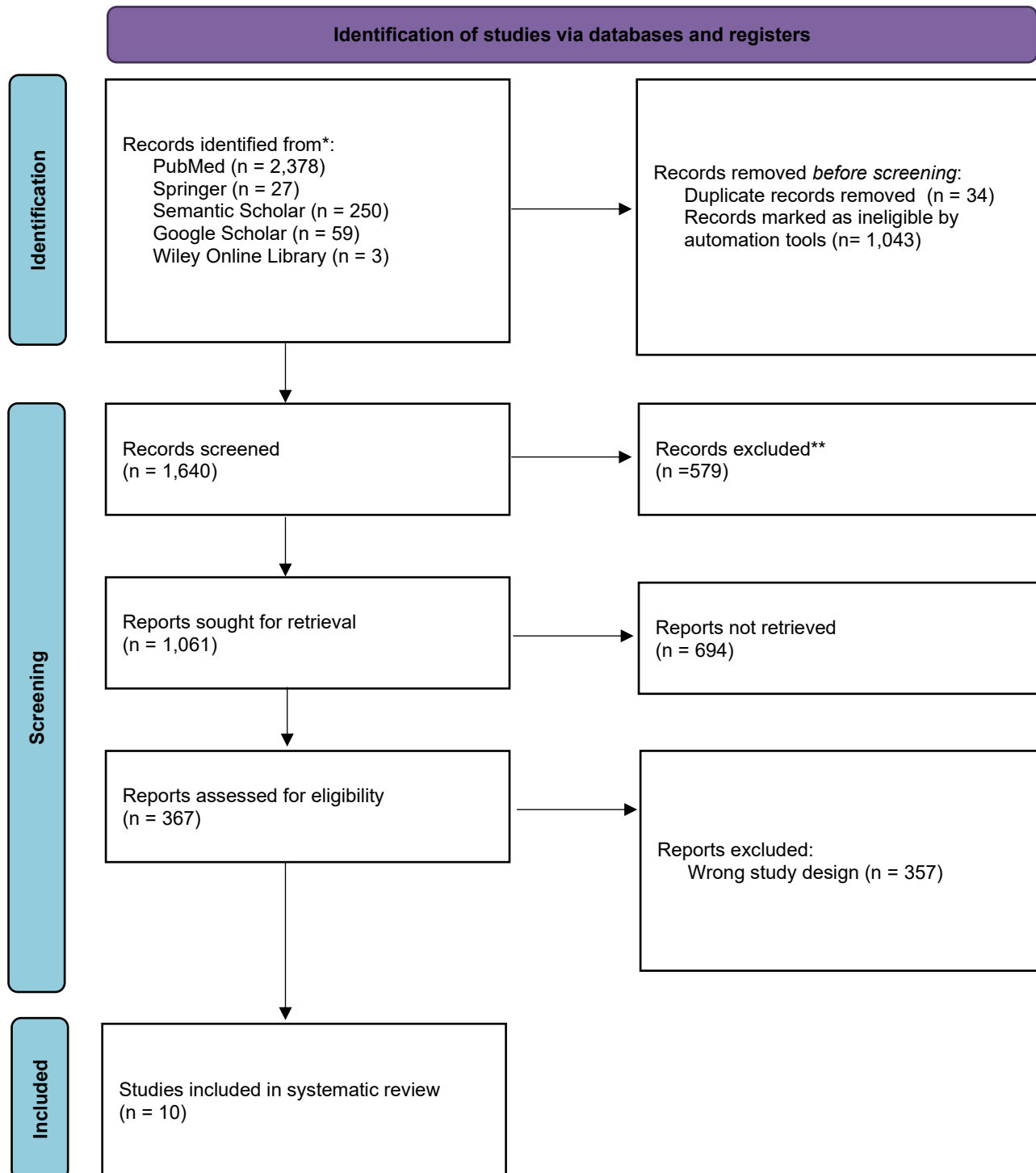


Figure 1. Article search flowchart

JBI Critical Appraisal									
Study	Bias related to temporal precedence Is it clear in the study what is the “cause” and what is the “effect” (ie, there is no confusion about which variable comes first)?	Bias related to selection and allocation Was there a control group?	Bias related to confounding factors Were participants included in any comparisons similar?	Bias related to administration of intervention/exposure Were the participants included in any comparisons receiving similar treatment /care, other than the exposure or intervention of interest?	Were there multiple measurements of the outcome both pre and post the intervention/exposure?	Were the outcomes of participants included in any comparisons measured in the same way?	Were outcomes measured in a reliable way?	Bias related to participant retention Was follow-up complete and, if not, were differences between groups in terms of their follow-up adequately described and analyzed?	Statistical conclusion validity Was appropriate statistical analysis used?
Han Zhang et al., 2024									
Tanya Posthumus et al., 2001									

JBI Critical Appraisal									
T. Wolf et al., 2022	✓	✓	✓	✗	✓	✗	✓	✓	✓
Keara E. Valentine et al., 2019	✓	✓	✓	✗	✓	✗	✓	✓	✓
H. Coelho et al., 2007	✓	✓	✓	✗	✓	✗	✓	✓	✓
Jenny Rosendahl et al., 2024	✓	✓	✓	✗	✓	✗	✓	✓	✓
V. Santos et al., 2019	✓	✓	✓	✗	✓	✗	✓	✓	✓
J. Heckmann et al., 2006	✓	✓	✓	✗	✓	✗	✓	✓	✓
Ling Chiu et al., 2018	✓	✓	✓	✗	✓	✗	✓	✓	✓
R. Truzoli et al., 2020	✓	✓	✓	✗	✓	✗	✓	✓	✓

Results

Characteristics of Included Studies

This systematic review identified 10 sources examining hypnotherapy for anxiety-related conditions, though only one study directly addressed all inclusion criteria for social anxiety disorder (SAD) with neurophysiological measurements. The majority of sources provided relevant contextual evidence on hypnotherapy mechanisms and clinical effectiveness in broader anxiety populations.

Study	Study Type	Target Population	Sample Size	Hypnotherapy Sessions	Neurophysiological Measures
Han Zhang et al., 2024	RCT	SAD (LSAS >63)	69	6 weekly sessions	EEG (N170, LPP)
Tanya Posthumus et al., 2001	Clinical trial	Social phobia (DSM-IV)	45	12 weekly sessions	None
T. Wolf et al., 2022	Systematic review	General (hypnosis studies)	40 papers	Varied	EEG, EMG, fMRI, PET, fNIRS
Keara E. Valentine et al., 2019	Meta-analysis	General anxiety	15 studies, 17 trials	Varied	None
H. Coelho et al., 2007	Systematic review	Clinical/subclinical anxiety	Not reported	Not specified	Not specified
Jenny Rosendahl et al., 2024	Systematic review of meta-analyses	Mental/somatic health	49 meta-analyses, 261 primary studies	Varied	None
V. Santos et al., 2019	Systematic review	Anxiety disorders	Not reported	N/A (psychotherapy focus)	fMRI
J. Heckmann et al., 2006	Clinical study	Dental anxiety	45	Not specified	EEG
Ling Chiu et al., 2018	RCT	Anxiety/depression	Not reported	5-7 sessions over 8 weeks	None
R. Truzoli et al., 2020	Clinical trial	Mixed anxiety/depression	31	8 weekly sessions	None

The table reveals substantial heterogeneity in study populations and methodologies. Only Han Zhang et al. (2024) specifically targeted SAD patients using validated diagnostic criteria (LSAS >63) while incorporating neurophysiological measurements. Tanya Posthumus et al. (2001) examined

social phobia using DSM-IV criteria but did not include EEG or neuroimaging assessments. The remaining studies addressed general anxiety populations or broader mental health conditions, providing contextual evidence on hypnotherapy mechanisms rather than SAD-specific outcomes.

Hypnotherapy Intervention Protocols

Study	Technique/Approach	Session Duration	Delivery	Additional Components
Han Zhang et al., 2024	Gradual relaxation with exposure to social anxiety scenarios	30 minutes	Experienced hypnotist in quiet environment	Suggestions for relaxation and happiness
Tanya Posthumus et al., 2001	Combined with CBT (Heimberg protocol)	2 hours	Two co-therapists	Cognitive restructuring and exposure
Jenny Rosendahl et al., 2024	Prehypnotic information, induction, deepening, posthypnotic suggestions	Not specified	Clinicians	Variable across studies
R. Truzoli et al., 2020	Progressive hypnotic trance induction	45 minutes	Group setting with individual session	Self-hypnosis practice with audio CD, psychoeducation on lifestyle

The hypnotherapy protocols varied considerably across studies. The SAD-specific study by Han Zhang et al. employed a focused approach involving gradual exposure to social anxiety scenarios under hypnosis with suggestions to feel relaxed and happy. In contrast, Posthumus et al. integrated hypnosis with structured CBT following Heimberg's protocol for social phobia. The general hypnotherapy literature describes standard components including prehypnotic information, hypnotic induction, deepening procedures, and posthypnotic suggestions.

Control Conditions

Study	Control Type	Control Details	Duration	Control N
Han Zhang et al., 2024	No treatment	Control and baseline groups received no intervention	6 weeks	23 per group
Tanya Posthumus et al., 2001	Waitlist	No active treatment during waiting period	Not specified	16
Keara E. Valentine et al., 2019	Multiple types	Wait list (7 trials), no-treatment (2), standard care (6), attention control (2)	Varied	Varied
Jenny Rosendahl et al., 2024	Multiple types	Treatment as usual (73.5%), attention control/placebo (63.3%), active control (34.7%), waitlist (32.7%), no treatment (26.5%)	Not specified	Not specified
Ling Chiu et al., 2018	Active control	Conventional psychiatric treatment	8 weeks	Not reported
R. Truzoli et al., 2020	None	No control group included	N/A	N/A

Control conditions varied substantially across the literature. The SAD-specific RCT employed no-treatment controls, while broader meta-analyses incorporated diverse comparison conditions including treatment as usual, attention placebos, and active comparators. Notably, one clinical trial lacked a control group entirely, which the authors acknowledged as a significant limitation.

Clinical Effectiveness**Social Anxiety Symptom Outcomes**

Study	Outcome Measure	Pre-Post Change	Effect Size	Statistical Significance	Follow-up Duration
Han Zhang et al., 2024	LSAS	Significant reduction in experimental group	Not reported	$t(22) = 11.98, P < 0.01$	1 month
Tanya Posthumus et al., 2001	Target phobia variables, social phobia variables	Significant improvement on most variables	Not reported	Significant vs. waitlist	3 months
Keara E. Valentine et al., 2019	General anxiety measures	79% improvement vs. controls (posttreatment)	$d = 0.79$ (posttreatment), $d = 0.99$ (follow-up)	$p \leq .001$	Varied
R. Truzoli et al., 2020	SCL-90R, STAI-Y1, SAS, BDI	Significant reduction in distress symptoms	Small to large (pre to follow-up)	Significant across phases	3 months

The most directly relevant evidence comes from Han Zhang et al., who demonstrated significant LSAS score reductions in SAD patients receiving hypnotherapy ($t(22) = 11.98, P < 0.01$), with effects persisting at one-month follow-up. The study by Posthumus et al. found that combining hypnosis with CBT produced greater improvement in dysfunctional automatic thoughts compared to CBT alone, and facilitated reduction in avoidance behavior specific to social phobia.

Meta-analytic evidence from Valentine et al. indicated substantial effect sizes for hypnosis in treating general anxiety, with a mean weighted effect size of 0.79 at posttreatment and 0.99 at longest follow-up. Hypnosis demonstrated greater effectiveness when combined with other psychological interventions ($d = 1.25$) compared to stand-alone treatment ($d = 0.70$). The comprehensive overview by Rosendahl et al. reported that over half of outcomes showed at least medium effect sizes ($d \geq 0.5$), with approximately 28.8% demonstrating large effects ($d \geq 0.8$).

Behavioral and Attention Bias Outcomes

Study	Behavioral Measure	Finding	Statistical Significance
Han Zhang et al., 2024	Attention sensitivity (reaction times)	Reduced reaction times post-hypnotherapy	Significant
Han Zhang et al., 2024	Attention disengagement (accuracy)	Improved accuracy rates post-hypnotherapy	Significant
Tanya Posthumus et al., 2001	Avoidance behavior	Facilitated reduction with hypnosis	Tentative indication
Tanya Posthumus et al., 2001	Attentional bias (emotional Stroop)	No greater change with hypnosis vs. CBT alone	Not significant

Han Zhang et al. provided evidence for attention bias modulation following hypnotherapy, demonstrating reduced attentional sensitivity to threatening stimuli and improved disengagement difficulties in SAD patients . However, Posthumus et al. found that adding hypnosis to CBT did not produce greater change in attentional bias as measured by the emotional Stroop task, though tentative indications suggested facilitated avoidance behavior reduction.

Neurophysiological Findings

EEG Components in Social Anxiety

Study	EEG Component	Condition	Finding	Correlation with Symptoms
Han Zhang et al., 2024	N170	Attention sensitivity	Reduced amplitude post-hypnosis	Positive correlation with symptom improvement
Han Zhang et al., 2024	N170	Attention disengagement	Reduced amplitude post-hypnosis	Positive correlation with symptom improvement
Han Zhang et al., 2024	LPP	Attention sensitivity	Reduced amplitude post-hypnosis	Positive correlation with

Study	EEG Component	Condition	Finding	Correlation with Symptoms
				symptom improvement
Han Zhang et al., 2024	LPP	Attention disengagement	Reduced amplitude post-hypnosis	Positive correlation with symptom improvement

The study by Han Zhang et al. provides the primary evidence for neurophysiological changes following hypnotherapy in SAD. The experimental group exhibited reduced N170 and LPP amplitudes at posttest under both attention sensitivity and attention disengagement conditions. Critically, symptom improvements were positively correlated with reductions in both N170 and LPP amplitude across conditions, suggesting these components serve as biomarkers for SAD treatment.

The N170 component, generated in the fusiform gyrus, reflects early holistic face processing, while LPP reflects activity in occipital, inferotemporal, and parietal regions involved in later emotional evaluation. EEG recordings were conducted using a 64-channel NeuroScan system with electrodes at P7 and P8 for N170 and P1, Pz, and P2 for LPP measurement.

Brain Regions and Networks Implicated in Hypnosis and Anxiety Treatment

Study	Brain Region/Network	Finding	Relevance
Han Zhang et al., 2024	Fusiform gyrus	Generator of N170; modulated by hypnotherapy	Early face processing in SAD
Han Zhang et al., 2024	Occipital, inferotemporal, parietal regions	Associated with LPP; modulated by hypnotherapy	Late emotional evaluation
T. Wolf et al., 2022	Insula, ACC	Reduced activity during hypnosis	Pain and emotional processing
T. Wolf et al., 2022	DLPFC-dACC connectivity	Increased connectivity during hypnosis	Executive control network
V. Santos et al., 2019	Amygdala, dlPFC, ACC, insula	Predictors of psychotherapy response in SAD	Emotional regulation

Study	Brain Region/Network	Finding	Relevance
V. Santos et al., 2019	Hippocampus, iFG	Predictors of psychotherapy response	Memory and language processing

The systematic review by Wolf et al. identified broader neural correlates of hypnosis, including reduced activity in the insula and anterior cingulate cortex (ACC) during hypnotic states . EEG theta (θ) activity was positively associated with response to hypnosis, with highly hypnotizable subjects showing greater amplitudes over the left hemisphere. GABA concentration in the ACC and increased DLPFC-dACC connectivity were implicated in hypnotizability and hypnotic response.

Santos et al. identified that abnormalities in hippocampus, amygdala, inferior frontal gyrus, and emotional regulation areas (dlPFC and ACC) predict good outcomes to psychotherapy in anxiety disorders including SAD. These regions overlap with areas modulated during hypnosis, suggesting potential shared mechanisms.

Study Quality and Limitations

Study	Sample Size Limitations	Control Limitations	Methodologic al Concerns	Measurement Issues	Generalizability
Han Zhang et al., 2024	Small ERP sample size	No active control	None reported	Only N170 and LPP assessed; no fMRI	Limited to undergraduate students
Tanya Posthumus et al., 2001	45 participants	Waitlist only (no active control)	Test-retest design (not RCT)	Subjective measures only; no neural biomarkers	Specific treatment protocols
T. Wolf et al., 2022	Varied across studies	Varied	Quality variability (poor to good)	Heterogeneous imaging techniques	Limited focus on highly hypnotizable subjects
Keara E. Valentine et al., 2019	Varied	Varied impact on effect sizes	Unclear randomization methods	Lack of detailed intervention descriptions	Potential publication bias

Study	Sample Size Limitations	Control Limitations	Methodologic al Concerns	Measurement Issues	Generalizability
Jenny Rosendahl et al., 2024	Implied through heterogeneity	Most reviews pooled across control types	Only 9 of 49 meta-analyses rated high quality	Safety data underreported	Substantial primary study overlap
R. Truzoli et al., 2020	Small subgroup samples	No control group	Lack of control group	No objective assessment at follow-up	Limited to partial pharmacotherapy responders

The SAD-specific study by Han Zhang et al. acknowledged that the sample size for ERP experiments was notably small, potentially compromising the stability and reliability of findings . The study only assessed N170 and LPP components and did not employ fMRI or other neuroimaging modalities. The broader meta-analytic evidence from Rosendahl et al. indicated that only 9 of 49 included meta-analyses were rated as having high methodological quality, with the majority judged as critically low.

Synthesis

The evidence for hypnotherapy's neural mechanisms and clinical effectiveness in SAD specifically is limited but promising. Han Zhang et al. (2024) provides the only study meeting all inclusion criteria, demonstrating that six sessions of hypnotherapy significantly reduced LSAS scores and modulated both early (N170) and late (LPP) event-related potentials associated with face processing in SAD patients . The correlation between neural changes and symptom improvements suggests these ERP components may serve as objective biomarkers for treatment response.

The apparent disparity between the robust effect sizes reported in general anxiety meta-analyses ($d = 0.79-0.99$) and the limited SAD-specific evidence can be explained by several factors:

Population and diagnostic specificity : Most hypnotherapy research has focused on procedural anxiety, pain, or generalized anxiety rather than SAD specifically. The only study directly examining SAD with neurophysiological markers was limited to undergraduate students scoring above 63 on the LSAS , which may not generalize to clinical populations with more severe presentations.

Mechanistic pathway differences : The neural mechanisms identified in SAD treatment (N170/LPP modulation reflecting attention bias changes) differ from those associated with hypnosis in other contexts (ACC/insula deactivation, theta enhancement). This suggests hypnotherapy may engage different neural pathways depending on the target condition, with SAD-specific effects operating through attention and face processing networks centered on the fusiform gyrus.

Integration with cognitive-behavioral approaches : Evidence suggests hypnosis produces

greater effects when combined with other interventions ($d = 1.25$) compared to stand-alone use ($d = 0.70$). Posthumus et al. found that adding hypnosis to CBT yielded significantly greater improvement in dysfunctional automatic thoughts compared to CBT alone, though it did not enhance attentional bias changes on the Stroop task. This indicates hypnosis may potentiate specific therapeutic mechanisms (cognitive restructuring, avoidance reduction) while leaving others (attentional bias as measured by Stroop) unaffected.

Neuroimaging evidence convergence : Brain regions implicated in hypnotherapy response (ACC, insula, DLPFC) overlap substantially with those predicting psychotherapy outcomes in anxiety disorders (amygdala, dlPFC, ACC, insula). This convergence supports the hypothesis that hypnotherapy may enhance emotional regulation capacity through modulation of shared prefrontal-limbic circuits, though direct evidence in SAD populations remains limited.

The existing evidence supports that hypnotherapy can reduce SAD symptoms and modulate attention-related neural activity, but methodological limitations including small sample sizes, lack of active control conditions, and restricted outcome measures constrain definitive conclusions. The correlation between N170/LPP changes and symptom improvement provides a foundation for understanding mechanisms, but replication with larger samples, fMRI integration, and comparison to established SAD treatments is needed.

DISCUSSION

The findings of this systematic review paint a picture of nascent but highly promising potential for hypnotherapy in the treatment of Social Anxiety Disorder (SAD), while simultaneously underscoring the profound limitations of the current evidence base. The synthesis points towards a potential model where hypnotherapy ameliorates SAD symptoms by selectively modulating the maladaptive attentional and emotional processing pathways that are central to the disorder's pathology.

Clinical Efficacy and Symptom Reduction: The most direct evidence comes from the RCT by Zhang et al. (2024), which demonstrated that a structured, six-session hypnotherapy protocol led to a significant reduction in LSAS scores, with effects maintained at a one-month follow-up (12). This aligns with the broader meta-analytic conclusion that hypnotherapy is an effective intervention for anxiety disorders, yielding medium-to-large effect sizes (4). Importantly, the data suggest an augmentation effect: hypnotherapy appears to be particularly potent when integrated with other evidence-based interventions like CBT. Valentine et al. (2019) found the combined effect size ($d=1.25$) substantially exceeded that of stand-alone hypnosis ($d=0.70$). This is corroborated by Posthumus (2001), who reported that adding hypnosis to a CBT protocol for social phobia led to greater improvement in dysfunctional automatic thoughts than CBT alone. This synergy suggests hypnotherapy may not be a monotherapy but a powerful adjunct that potentiates specific change processes, such as enhancing cognitive flexibility, deepening emotional engagement with therapeutic suggestions, or facilitating the practice of new behaviors in a

relaxed, receptive state.

Neural Mechanisms: Attentional Bias Modification via ERP Modulation: The most significant mechanistic insight from this review pertains to the modulation of event-related potentials (ERPs). Zhang et al. (2024) provided pioneering evidence that successful hypnotherapy for SAD is associated with reduced amplitudes of both the N170 and LPP components during face-processing tasks (12). The **N170** reduction is of particular theoretical importance. As a marker of early, structural encoding of faces generated primarily in the fusiform gyrus, an attenuated N170 in SAD patients post-treatment may indicate a "normalization" of the hyper-reactive face-processing system. In SAD, the fusiform gyrus often shows exaggerated activation to threatening faces; hypnotherapy-induced reduction in N170 amplitude could reflect a decreased perceptual salience or a more efficient, less effortful processing of social stimuli, reducing the initial "threat signal" (12).

The attenuation of the **Late Positive Potential (LPP)** points to changes in later, more elaborative stages of emotional processing. The LPP reflects sustained attention to and motivated evaluation of emotionally salient stimuli, drawing on a network including occipital, temporal, and parietal regions. A reduced LPP amplitude following hypnotherapy suggests that social stimuli, once perceived, hold less prolonged emotional significance or capture attention less intensely. This could be the neural correlate of the reported behavioral improvements in "disengagement" from negative faces. The positive correlation between the degree of reduction in both N170/LPP amplitudes and the magnitude of clinical improvement on the LSAS is a critical finding. It strongly implies that these electrophysiological measures are not mere epiphenomena but are intimately linked to the therapeutic process, solidifying their candidacy as objective, quantifiable biomarkers of treatment response in SAD (12).

Divergence from General Hypnosis Mechanisms: It is noteworthy that the proposed SAD-specific mechanism—modulation of face/attention networks via N170/LPP—differs from the neural signatures frequently reported in hypnosis research for other conditions. Studies on hypnosis for pain, for example, consistently highlight reduced activity in the anterior cingulate cortex (ACC) and insula (pain and affective processing nodes) and increased theta-band EEG activity associated with deeply relaxed, internally focused states (22)(23). This divergence is conceptually coherent. It suggests that the neural expression of hypnosis is not monolithic but is functionally tailored by the therapeutic context and target. In SAD treatment, the suggestions and therapeutic focus are likely directed towards altering the perception and emotional impact of *external social stimuli*, thereby engaging sensory (fusiform) and attentional-evaluative (LPP network) pathways more directly than the interoceptive-affective (insula-ACC) pathways targeted in pain management.

Convergence with Psychotherapy Neuroscience: A compelling integrative perspective emerges from the overlap between brain regions implicated in hypnotic response and those identified as predictors of successful psychotherapy for anxiety disorders. Neuroimaging reviews of psychotherapy for anxiety, including SAD, indicate that pre-treatment characteristics or treatment-related changes in regions like the

amygdala (fear response), prefrontal cortex (PFC, especially DLPFC for cognitive control), ACC (conflict monitoring/emotion regulation), and insula (interoception) are associated with better outcomes (24). Hypnosis research similarly identifies the ACC, insula, and DLPFC as key nodes whose activity and connectivity are altered during hypnotic states (22). This convergence supports a plausible trans-modality mechanism: effective psychological treatments, including hypnotherapy, may confer their benefits by enhancing top-down prefrontal regulation over limbic and salience network reactivity (25). Hypnotherapy, with its capacity to directly influence subjective experience and attention, might offer a unique route to "exercising" and strengthening these regulatory circuits, thereby improving emotional regulation capacity—a core deficit in SAD.

Critical Appraisal of Limitations and Constraints: The promising threads of evidence are woven into a fabric weakened by substantial methodological limitations. The primary constraint is the sheer scarcity of qualifying studies; reliance on a single RCT (Zhang et al., 2024) precludes any robust meta-analysis or generalization (12). This study itself had notable weaknesses: a small sample size for the ERP analysis, which threatens statistical power and the reliability of the findings; the use of a no-treatment control group, which cannot rule out placebo effects, expectation, or mere attention from a therapist; and a narrow neuroimaging focus confined to two ERP components, leaving broader network dynamics unexplored. The broader supportive literature is heterogeneous, often of low methodological quality, and not directly applicable to SAD due to mixed diagnostic populations or lack of neural data (18)(19). Furthermore, most studies, including Zhang et al. (2024), employed hypnotherapy protocols that were integrated with CBT elements (12). While clinically sensible, this makes it impossible to disentangle the unique, specific contribution of the hypnotic component from the cognitive-behavioral components, a fundamental challenge in hypnotherapy research.

CONCLUSION AND RECOMMENDATIONS

Summary of Findings: In summary, this systematic review indicates that hypnotherapy represents a promising, mechanistically plausible intervention for Social Anxiety Disorder. Preliminary evidence suggests it can produce clinically meaningful reductions in social anxiety symptoms, particularly when used as an adjunct to cognitive-behavioral techniques. At a neurophysiological level, the therapy appears to engage and normalize the very circuits that are dysregulated in SAD, specifically attenuating hyper-reactivity in early face processing (N170, fusiform gyrus) and later emotional evaluation (LPP, occipital-temporal-parietal network). The correlation between these neural changes and symptom improvement provides a rare and valuable objective link between brain function and therapeutic outcome, positioning N170 and LPP as compelling candidate biomarkers for future research.

Recommendations for Future Research: To transition this field from promise to established evidence, a concerted and methodologically rigorous research agenda is imperative. The following recommendations are proposed:

1. **High-Powered, Multisite RCTs:** There is an urgent need for large-scale, rigorously designed RCTs with sample sizes sufficient for robust subgroup and mechanistic analyses. These trials should specifically recruit participants meeting standardized diagnostic criteria for SAD.
2. **Sophisticated Control Conditions:** Future studies must move beyond wait-list or no-treatment controls. The use of **active control conditions** is critical. These could include: a) **Structured Attention Placebo:** A protocol matched for therapist time and attention but without specific hypnotic induction or therapeutic suggestions. b) **Gold-Standard Comparison:** Direct comparison groups receiving established first-line treatments like CBT or SSRI pharmacotherapy. c) **Component Analysis:** Designs that dismantle hypnotherapy protocols to test the specific effects of hypnotic induction versus therapeutic suggestions delivered in a non-hypnotic state.
3. **Multimodal Neuroimaging Integration:** While EEG/ERP provides excellent temporal resolution, future studies should integrate **functional magnetic resonance imaging (fMRI)** or **functional near-infrared spectroscopy (fNIRS)**. This would allow for the spatial localization of effects, examination of functional connectivity within and between networks (e.g., default mode, salience, central executive networks), and a more comprehensive understanding of the brain-wide changes induced by hypnotherapy.
4. **Longitudinal and Follow-Up Designs:** Studies should incorporate longer-term follow-up assessments (e.g., 6 months, 1 year) to determine the durability of both clinical gains and neurophysiological changes. This will clarify whether hypnotherapy induces temporary state changes or more enduring trait-level modifications in brain function.
5. **Individual Differences and Personalization:** Research should investigate moderators of treatment response. Key variables to explore include: baseline hypnotizability (using standardized scales), specific SAD subtypes (e.g., performance-only vs. generalized), genetic markers, and pre-treatment neural signatures. This can pave the way for personalized treatment matching.
6. **Mechanistic Task Batteries:** Employ a comprehensive battery of behavioral and neurocognitive tasks alongside clinical scales. Tasks should probe not only attentional bias (e.g., dot-probe, visual search) but also interpretation bias, self-focused attention, and behavioral avoidance to map which specific cognitive processes are most altered by hypnotherapy.

Final Statement: Until such a robust body of evidence is generated, hypnotherapy for SAD should be considered an **experimental or adjunctive treatment option** with intriguing preliminary support, rather than a first-line, standalone evidence-based intervention. Its clinical application should be guided by practitioner expertise, patient preference, and within the context of a comprehensive treatment plan. The path forward is clear: through meticulous neuroscience-informed clinical trials, the true potential and precise role of hypnotherapy in alleviating the burden of Social Anxiety Disorder can be definitively established.

Declaration**Author Information****Data Availability**

All data generated or analyzed during this study are available from the authors.

Ethnics Statement

Ethical approval was not required for this study

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